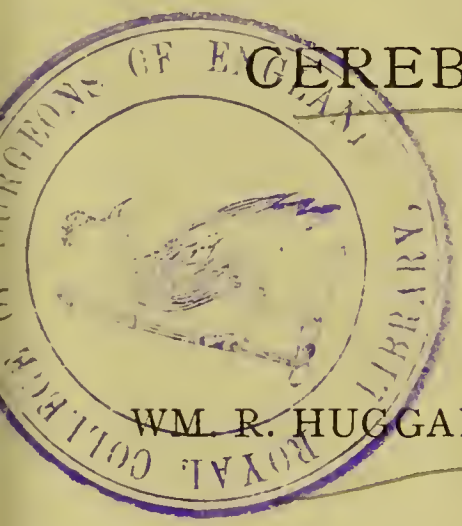
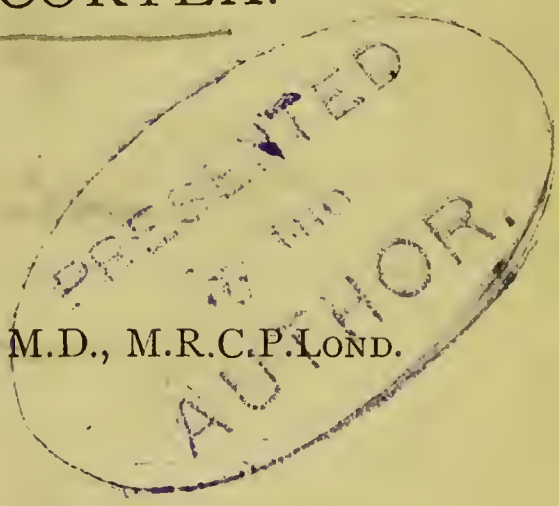
# PROFESSOR SCHIFF'S EXPERIMENTS

ON THE

# EXCITABLE AREA OF THE CEREBRAL CORTEX.

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*Reprinted from* The Lancet *of August* 1, 1885.

# PROFESSOR SCHIFF'S EXPERIMENTS ON THE EXCITABLE AREA OF THE CEREBRAL CORTEX.

PROFESSOR SCHIFF has kindly given me permission to report some of his hitherto unpublished experiments on the functions of the excitable area of the cerebral cortex. Most of these experiments I have myself witnessed. It is known that Professor Schiff regards this portion of the brain as presiding, not over motility, but over tactile sensibility.[1] He has recently devised some new experiments that tend strongly to confirm his view. It is to be understood that the following remarks have reference only to such animals as dogs, cats, rabbits, rats, &c. Monkeys yield results in many respects different.

The doctrine ordinarily accepted, in England at any rate, is that the excitable area of the cortex is motor, or perhaps psycho-motor, in function. The grounds on which this belief is based are that the irritation with a weak electric current of various definite parts of this area give rise to definite and localised movements on the opposite side. Within certain limits the movements are the same when the same spot is irritated; and precise spots for the various groups of movements have been mapped out. If this

[1] See L'Imparziale Med. di Firenze, 1871.

portion of the cortex be removed, some of the movements of the animal are performed with less certainty on the side opposite the lesion. When the animal has quite recovered from the wound, if the same part of the brain be again exposed, irritation no longer gives rise to movement. From these facts, and from kindred experiments on monkeys, it has been inferred that the excitable spots in the cortex are motor centres. The facts on which the inference is based are unimpeachable; but they do not warrant the inference. The facts are true, but they are not the whole truth. Professor Schiff's experiments show, not merely that an important element, but that the essential factor, has been overlooked. These experiments establish the following points: (1) that the removal of the excitable area of the cortex, or so-called motor region, permanently abolishes tactile sensibility; (2) that it does not diminish either sensibility to pain or motor power, strictly so-called; (3) that the abolition of tactile sensibility is sufficient to account for the alteration in the animal's motility; (4) that the centres of nutrition of the fibres connecting the excitable area of the cortex with the true motor centres are in the ganglia of the posterior roots of the spinal nerves, and that the fibres in question are in physiological continuity with the posterior columns of the cord; (5) that when central[2] tactile sensibility is abolished the so-called motor region is no longer excitable. The gist of the evidence in support of these points will now be stated concisely.

On May 8th an opening was made with a trephine over the left crucial sulcus of a dog. The application of a weak induced current showed that the excitable area was reached. The whole of the excitable area—namely, the sigmoid

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[2] Compare Schiff: Lehrbuch der Muskel und Nerven Physiologie, 1858, p. 216.

gyrus, with part of the convolution behind—was then removed. When the animal had recovered from the anæsthesia it ran about as if nothing were amiss. On the smooth floor the right limbs showed some ataxia and uncertainty of movement, with tendency to slip—an appearance that might at first sight seem to be due to muscular weakness. On a rough surface, however, this ataxia, uncertainty of movement, and slipping altogether disappeared, and in running and jumping the animal was quite indistinguishable from a healthy dog. The so-called motor centres had been removed, but motility had not been interfered with, or, rather, it was interfered with only when the animal was on a smooth surface. What was the explanation of this? We shall see presently.

Tactile sensibility was tested and was found to be absent. A clamp with weak spring, to avoid painful pressure (which does not belong to the same class of sensations as touch), was applied to various places on the left side, and on every occasion the animal showed signs of impatience, and shook it off. When it was applied to the right side no notice whatever was taken of it. For example, when the clamp was fixed to the left nostril, lip, or ear, the dog shook its head violently, and so got rid of the offending body. When it was put on the web between the toes of either the fore or hind paw of the left side, the animal either shook it off or removed it with its head. On the corresponding parts of the right side the clamp was quite unnoticed. A current of air, too, on the left ear was sharply resented; on the right ear it was disregarded. On the right side, moreover, when the toes were doubled up, the animal stood without observing the position of the limbs. Sensibility to pain was unaltered. The prick of a pin gave rise to identical phenomena on both sides. On the right side, as on the left, the limb was quickly

withdrawn from the pin. After a month the condition was still the same with regard to both varieties of sensation.

The explanation of the slipping, uncertainty of movement, and ataxia is now before us. These phenomena were due merely to loss of tactile sensibility. How could loss of tactile sensibility produce them? The numerous points of support in a rough surface prevented slipping. In the absence of these points slipping could only be avoided by a more delicate balancing of the body. But the very mechanism by which balance is adjusted was thrown out of gear. Hence the uncertainty of movement and ataxia. Loss of tactile sensibility was proved to be present; but it may be thought that loss of tactile sensibility is not enough to account for the phenomena. Experimental proof, however, is at hand. The phenomena were precisely the same, as far as the limbs were concerned, in two dogs, when the posterior column was cut on one side in the cervical region of one dog, and when in the other the excitable area of the cortex on the opposite side was removed. In both cases tactile sensibility was the only function that could be shown to be lost; and in both cases the same form of ataxia—ataxia on a smooth surface—was present. A striking variation of this experiment was made. The excitable area of the cerebral cortex of a dog was removed on one side, and three weeks later the posterior column of the cord in the cervical region was cut on the same side. Apart from traumatic effects, the results in the limbs were identical on both sides; and three weeks after the second operation the only difference between the two sides was that on the side opposite the cortical lesion tactile sensibility was lost in the face as well as in the limbs.

The crowning evidence, however, that the excitable area of the cortex is the area of tactile sensibility is yet to come.

In dogs whose posterior columns were cut in the cervical region, if after the lapse of about four days the so-called motor region is laid bare, it is found on both sides to be no longer excitable. If one posterior column only is cut, the opposite side of the brain only is inexcitable. If the posterior columns or posterior roots are cut in the lumbar region, irritation of the cortex still produces movements of the anterior limbs, but not of the posterior.

What is the explanation of this circumstance? Professor Schiff has found by numerous experiments that the disappearance of excitability takes place, not immediately, but on the fourth day after the cutting of the posterior columns. Albertoni discovered a kindred fact in regard to the disappearance of excitability after the ablation of the excitable cortex. The disappearance does not take place at once, but within about four days after the removal of the grey matter. Now, the period that elapses before degeneration, when a nerve is cut from its nutritive centre in the dog, is about four days. The significance of the fact is apparent. It is evident that when the excitable cortex is removed, and when the posterior columns or posterior roots are cut, there is equally a degeneration of the fibres that lead from the excitable area to the true motor centres. The inference is that these fibres are in physiological continuity with the posterior roots, where their nutritive centres are. But the fibres of the posterior roots are sensory fibres, and the fibres of the posterior columns are tactile sensory fibres.

It will, perhaps, be said that this argument proves too much; that it would prove that the fibres going from the excitable area to the true motor centres convey tactile sensibility—that is, convey sensory impressions to a motor centre. The facts as stated, however, do not involve this

supposition. The fibres connecting the area of tactile sensibility with the motor centres would transmit that sort of impression that causes a motor discharge. In other words, the grey matter of the excitable area transmutes sensibility into a liberating (probably voluntary) current.

Confirmatory evidence is found in the fact that in young animals the so-called motor centres are not excitable until the animal has learnt to coördinate its movements. In other words, until an association has been formed between tactile sensibility and suitable motions, stimulation of the area of tactile sensibility (so-called motor area) does not send any liberating current to the motor centres. The period of commencing excitability of the cortex is found to vary in different animals according to the period of commencing voluntary movement. Thus in cats and dogs the so-called motor centres are not excitable before the fifth day, while in guinea-pigs they are excitable on the second day. (Tarchanoff.) That the channels for outgoing impulses are already in working order before this is shown by the fact that powerful diffused currents produce general movements. The behaviour of the excitable region under anæsthesia is to the same effect. In an animal recovering from profound anæsthesia the so-called motor centres are found to be inexcitable, though general movements can be caused by currents strong enough to produce pain. Now, it is known that after anæsthesia tactile sensibility returns later than sensibility to pain. The obvious inference is that when tactile sensibility is gone the excitable area no longer responds to stimulation, even though motility and sensibility to pain are still preserved.

It is needful to say a word to prevent confusion between cortical excitability and the excitability of distant parts stimulated by the diffusion of powerful currents. Very weak currents suffice to elicit the definite and localised move-

ments that characterise the stimulation of the various parts around the *sulcus cruciatus*. Powerful currents, on the contrary, are diffused, and excite, probably through the pain-centre, general reflex movements. This distinction, though of capital importance, has not always been borne in mind by investigators, and disregard of it has more than once led to error.

If the so-called motor centres are not really motor centres, why, it may be objected, does stimulation of them give rise to movement? The answer is, that excitability, so far from proving them to be motor centres, tends to prove the reverse—to prove that they are sensory; for no other motor centres are known to be directly excitable. So far as has yet been ascertained, motor centres can be excited only through sensory nerves. The grey matter of the cord, for example, is absolutely inexcitable, according to almost all observers.

One of the greatest difficulties in experimental inquiry is the plurality of causes. This source of error vitiates most of the hitherto recorded researches on the present subject. A cause, lack of tactile sensibility, has been overlooked; and this cause has been demonstrated by Professor Schiff not merely to be present but to be sufficient to account for all the phenomena observed. This oversight on the part of skilful and able experimenters appears to have been due to dropping out of sight the fact that tactile sensibility is different from sensibility to temperature, from sensibility to pain, and from sensibility to pressure; and that its seat and channels of conduction are probably different also.

The whole matter may be summed up in a few words. The excitable area of the cortex in the dog is the area of tactile sensibility, and of this only. The evidence is as follows. 1. Ablation of the area in question permanently abolishes tactile sensibility on the opposite side; sensibility

to pain and motility being preserved. 2. The same symptoms, excluding the effects of traumatism, are produced by section of one of the posterior columns, and by removal on the opposite side of the excitable area for the limbs. In removing the excitable area care must be taken not to go deeper than the cortex, otherwise the true motor centres are reached. 3. The abolition of tactile sensibility by cutting the posterior columns of the cord abolishes the excitability of the so-called motor area, when time has been allowed for degeneration of the nerve fibres. 4. When tactile sensibility is annulled by anæsthesia, the excitable area is no longer excitable, even though movements can still be excited by pain.

Geneva.